Motor programmes for the termination of gait in humans: organisation and velocity-dependent adaptation

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1. The organisation of the muscular activities responsible for the termination of gait, their modulation as a function of the rate of progression and the associated mechanical effects were investigated in normal adults, using EMG, force plate and kinematic recordings. In particular, the braking actions in reaction to a visual cue presented at the instant of heel-strike were analysed quantitatively, with a focus on representative leg and thigh muscles of the weight-supporting (stance) and oscillating (swing) limb, during walk-and-stop trials performed at three different velocities.

2. In the stance limb, the EMG associated with braking started approximately 150 ms after the stop signal and, on average, displayed a distal-to-proximal activation sequence that primarily involved the posterior muscle groups (soleus, SOL, and hamstring, HAM). With the exception of SOL, which showed a single EMG burst, EMG patterns consisted of two or three progressively larger components occurring reciprocally in antagonistic muscles. Increasing walking speed yielded a significant reduction of the activity in distal muscles, and a simultaneous increment in proximal muscles. The mechanical effect of the earlier braking actions, estimated from the backward-directed wave of the horizontal ground reaction force, decreased in a velocity-dependent manner.

3. In the swing limb the braking activities began approximately 330 ms after the stop signal and, on average, revealed a proximal-to-distal activation sequence with the extensor groups (quadriceps, QUAD, and SOL) playing a prominent role. They always consisted of single EMG bursts, largely co-activated in the antagonist muscles. The onset latencies of the individual components showed a close correlation, and the spatio-temporal parameters were always scaled in parallel. Unlike the stance limb, the mechanical braking action associated with the final contact of the swing limb increased with walking speed.

4. The results indicate that the muscle synergies responsible for the rapid termination of gait in response to a ground-contact visual cue are produced by a relatively flexible set of motor commands modulated according to different velocity-dependent strategies in the weight-bearing limb, and by a single, fairly robust motor programme in the swing limb. Mechanical constraints related to the relative position of the centre of foot pressure and centre of body mass at the time the braking commands begin to affect external forces, may condition the difference between the two sides of the body.

Most of the available information on the physiology of locomotor control comes from studies performed under steady-state walking conditions. Among non-stationary locomotor behaviours, the initiation of gait has been examined in some detail (Carslöo, 1966; Herman *et al.* 1973; Brénière *et al.* 1981; Brénière & Do, 1987; Crenna *et al.* 1990; Crenna & Frigo, 1991*a*; Lepers & Brénière, 1995; Halliday *et al.* 1998). However, the opposite motor task (i.e. the transition from constant-speed walking to static postural attitudes) has received little attention. Investigation of the neural basis of the termination of gait in freely moving animals has shown that the activation of central areas located close to those involved in gait onset (e.g. the dorsal tegmental field of the caudal pons) can slow down and eventually stop spontaneous walking (Mori *et al.* 1989). The effects were ascribed to a

partial withdrawal of descending facilitatory drives to the antigravity muscles and to active inhibition of the propulsive activity (Mori, 1987).

Indirect insight into the mechanisms underlying the termination of gait in humans was obtained by measuring biomechanical variables such as ground reaction forces and relative trajectories of the centre of body mass (CM) and the centre of foot pressure (CP). The results of such studies demonstrate the contribution of 'negative' effects, reflected in the depression of the vertical and forward-directed ground reaction forces during propulsive phases, and 'positive' braking phenomena, evidenced by increased vertical and backward-directed reaction forces during body weight loading (Yamashita & Katoh, 1976; Jaeger & Vanitchatchavan, 1992). In the presence of favourable foot positioning, these two complementary actions cause displacement of the CP ahead of CM, leading to deceleration of the body (Jian *et al.* 1993). By means of a different protocol, whereby walking was abruptly interrupted in response to a strong cutaneous stimulation (to simulate hitting an obstacle), Hase & Stein (1998) obtained EMG confirmation of the intervention of negative and positive mechanisms.

According to the current notion, therefore, the termination of human gait is achieved by switching off the ongoing locomotor output, but it can also involve various excitatory actions that are bound to interfere with the current movement. In this respect the braking commands appear to operate as discrete self-paced perturbations, particularly at higher walking speeds, on account of the acceleration-dependent inertial load to be counteracted and the inherent unsteadiness of bipedal progression (Adamovich *et al.* 1994; Taga, 1995).

The characteristics of this commonplace motor task, entailing potentially destabilising interactions between voluntary (stopping) and mainly automatic (locomotor) commands, render the termination of gait an interesting topic in the field of motor control physiology, and raise a number of basic issues that have not been dealt with before. In the first place there is an obvious need for understanding in some detail the spatial and temporal organisation of the muscle activities responsible for deceleration in the weight-supporting limb and the swing limb. The modalities of interaction between the excitatory actions and the ongoing locomotor rhythm need to be elucidated, as do the associated mechanical effects. Moreover, the strategies adopted by the nervous system for tuning the braking commands in relation to walking speed still await analysis.

The present study sought to address these questions in order to provide a functional characterisation of the motor programmes responsible for the termination of human gait in one of the most natural conditions, i.e. in response to visual cues.

METHODS

Subjects

Five healthy volunteers participated in the experiments after giving their written informed consent. Their mean age, body mass and height was 32 years (range 19–45 years), 67 kg (range 62–74 kg) and 1.73 m (range 1.71–1.76 m), respectively. The local ethics committee gave approval for the study, and the procedures involved conformed to the Declaration of Helsinki.

Experimental set-up

Surface EMGs were collected from four representative lower limb muscles on both sides: soleus (SOL), tibialis anterior (TA), medial hamstrings (semimembranosus–semitendinosus complex; HAM) and quadriceps (vastus medialis; QUAD). The erectores spinae at the level of L3–L4 (ES) and the gluteus medius (GLM) were also recorded in three subjects. Electrode sites were prepared by cleansing the skin with alcohol and shaving hair, when necessary, to ensure good contact. Bipolar Ag–AgCl electrodes (interelectrode distance 25 mm, diameter 8 mm) were attached using adhesive pads and electrode gel. Flexible electrogoniometers (Penny and Giles, Gwent, UK) were fixed with double-sided tape across the hip and knee joints of both sides for the analysis of flexor–extensor angles. Recordings were taken in a gait laboratory equipped with a linear pathway (8 m long and 1.5 m wide) that allowed the subjects to achieve a steady walking rhythm in the central portion, where a force plate was embedded in the floor. This platform had the shape of an equilateral triangle (2 m each side) and included three sets of strain gauges, one at each corner, which measured the ground reaction forces on the three axes, and the centre of foot pressure (CP; Brénière & Do, 1986). Analog EMG signals were amplified (×1000), bandpass filtered (3–500 Hz), and the output of the EMG, force plate and goniometers was digitised at a sampling frequency of 1000 Hz (CED1401+ system; CED, Cambridge, UK) and saved on computer for further analysis.

Protocol

Subjects walked barefoot. Before data acquisition they performed several practice runs to become familiar with the recording set. In the first series of experimental trials, reference EMG, force plate and angular data were collected during steady walking at 'slow', 'natural' (preferred) and 'fast' speeds. The distance from the starting position on the walkway to the force plate was adjusted so that subjects always hit the platform with the right foot (Fig. 1, left panel). Ten control trials were recorded for each speed. In a second series of trials subjects were asked to walk at slow, natural and fast speeds, and terminate gait as soon as a white light was turned on. This stop signal was positioned at the end of the walkway, at the height of the subject's head, and was triggered by the right limb making contact with the force plate (maximum mechano-electrical delay 5 ms). During preliminary experiments when no constraints were imposed about the termination mode, subjects produced rapid stopping by adopting (1) a basic pattern, whereby the right limb started a moderate braking action and the contralateral limb added deceleration during the next ground contact, so that they stopped with the right limb backward and the left forward (stopping in one step), or (2) a more elaborate pattern, whereby the above sequence was followed by an additional short step by the right limb (stopping in one stride; see also Hase & Stein, 1998). When walking at slow speed, all subjects were able to stop in one step, whereas at natural and fast speeds, because of the higher residual velocity after the left foot made contact, they stopped in one stride in 30% and 98% of trials, respectively. Therefore, stopping in one stride was chosen for the present study, in order to compare braking activity across a full spectrum of functional walking speeds, using a single experimental protocol common to them all. To simplify comparisons, the length of the final step with

the right limb was kept constant for all walking speeds by asking subjects always to bring the right foot to a stop beside the contralateral foot (Fig. 1, right panel). The halt signal was presented randomly; out of 20 trials for each walking speed, 10 were blank and 10 were stop trials.

Data processing

Raw myoelectric signals were checked preliminarily for the presence of artefacts and crosstalk. Selected tracings were full-wave rectified, smoothed (80 Hz, zero phase shift, low-pass filter) and data recorded during constant-speed walking were averaged from 500 ms before the right limb heel-strike, to 2500 ms afterwards. As a result, three reference EMG templates (slow, natural and fast walking) and their standard deviations (S.D.) were stored for each muscle and for each subject. The stop trials were then analysed over the same time window. The EMG profiles recorded at the termination of gait were superimposed onto the corresponding reference templates, in order to isolate the positive effects, i.e. the extra EMG activity, and the negative effects, i.e. the actual decrements of the current locomotor output. The net positive braking components, obtained by subtraction, were examined in detail, provided their amplitude exceeded the reference profile by one S.D. for more than 100 ms. Their incidence is expressed as a percentage occurrence rate. Their onset and offset latencies with respect to the stop signal, and their durations, were determined from the times at which the actual EMG level crossed the reference profile by one S.D. Peak latencies were also measured. Timing parameters are expressed as absolute (milliseconds) and relative values (percentage of the duration of the stride cycle measured during steady walking at the corresponding speed). Peak amplitudes were computed and normalised to the maximum locomotor output detected on the reference EMG profile of the same muscle, at matched speed. For the SOL muscle, whose behaviour differed on the right and left side, the integrals of the positive braking components and of the background locomotor activity were also obtained. Averages $\pm$ S.D. of the EMG variables were computed for each class of speed and for all three walking speeds. Changes in these values in individual subjects as a function of the walking speed were further evaluated by Pearson's correlation coefficients and by the regression coefficient (slope), with statistical significance set at $P < 0.05$. In order to provide an overall estimate of the functional importance of each individual braking component, a 'consistency index' was finally computed, as follows: (% occurrence rate × % peak amplitude × % duration) × 10^{-5}.

Signals from the dynamometric platform and goniometers were filtered (80 Hz, zero phase shift, low-pass filter). The time courses of the ground reaction forces in the antero-posterior (Rx), lateral (Ry) and vertical (Rz) directions, and the instant position of the CP were

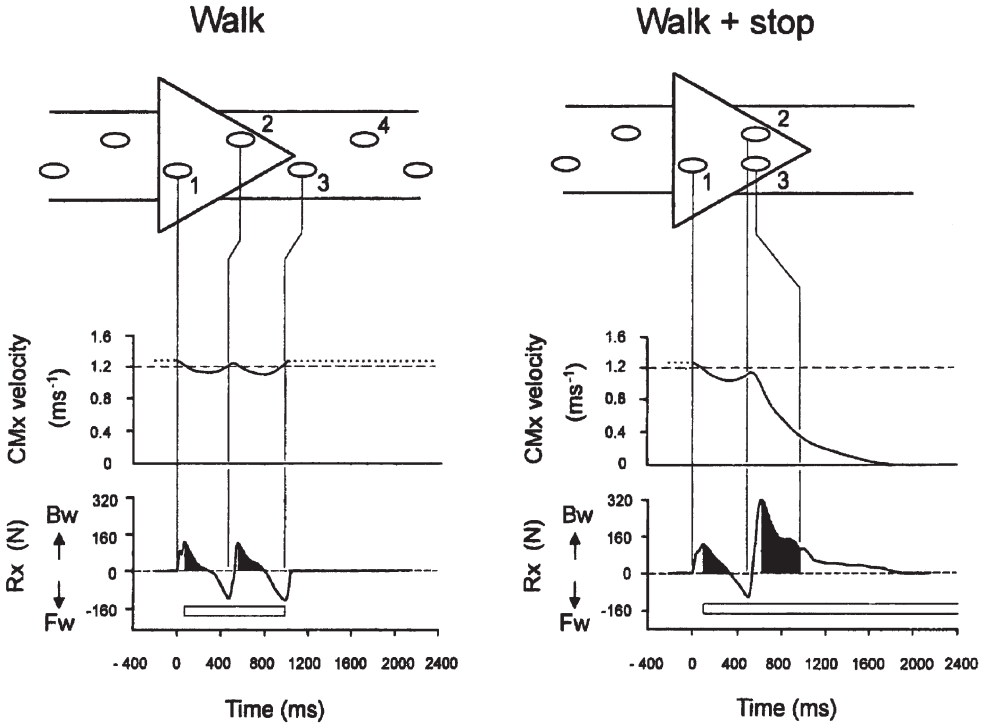

Figure 1. Patterns of foot placement and mechanical variables recorded during steady walking (Walk) and termination of gait (Walk + stop)

The upper panels give an overhead view of the walkway, triangular force plate and foot positions (walking direction from left to right). The vertical lines from the footprints mark the ground contact instants on the time courses of the horizontal velocity of the centre of body mass (CMx), and horizontal ground reaction forces (Rx), reported below. In the Rx profiles, the backward-directed (Bw) 'braking' components and the forward-directed (Fw) 'propulsive' components are represented as positive and negative waves, respectively. The shaded areas identify the single-limb support sub-periods that were selected for measuring the mechanical braking actions. Open boxes at the bottom mark the periods of full body-weight loading on the platform.

used for measuring stride dimensions and calculating the mean walking speed during steady walking trials (Bril & Brénière, 1992). Rx profiles and each subject's mass were employed for computing the accelerations of the centre of body mass (CM) in the corresponding direction, by the direct dynamics principle (see Brénière & Do, 1986). In the stop trials, where the acceleration of CM finally came to zero, integration of the acceleration signal on the x-axis gave the velocity of CM over the period when subjects loaded their whole body weight onto the platform (Fig. 1, lower panels, CMx). Analysis of the backward-directed component of Rx provided a quantitative estimate of the positive mechanical braking action in the post-contact phase in the control and stop trials. The area under the backward Rx wave recorded at the termination of gait was measured and normalised to the corresponding reference value. The braking actions produced on one side were assessed selectively by measuring the above areas during single-support periods (Fig. 1, lower panels, Rx, shaded areas). Biomechanical parameters were analysed statistically in the same way as EMGs.

RESULTS

In the gait-termination trials all muscles showed clear changes in the EMG profiles compared to steady walking. These consisted of positive events marked by the recruitment of extra activity, followed by negative events, involving the attenuation or even abolition of the current locomotor output. The positive events in the leg and thigh muscles, which were the main focus of the present study, included: (1) earlier components on the side that was going to bear the full body weight at the appearance of the stop signal (the right limb, hereafter

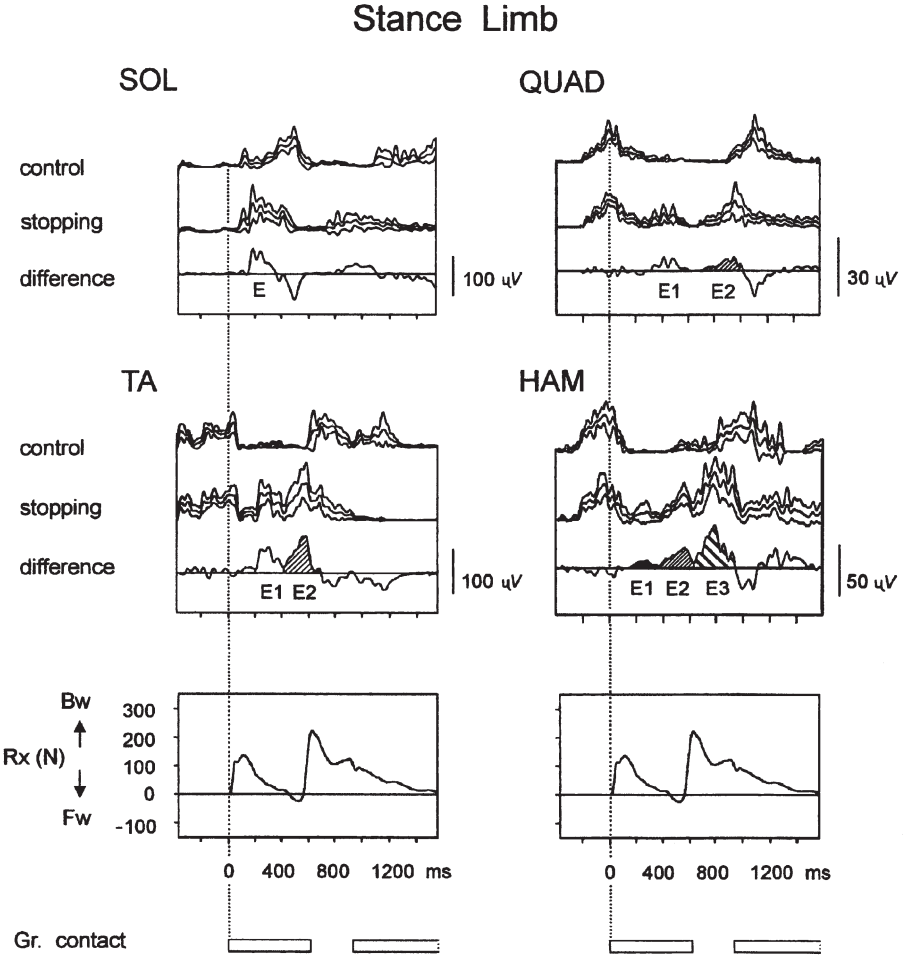

Figure 2. Positive braking activities in antagonistic leg and thigh muscles of the stance limb in a representative subject

For each muscle, the upper trace (control) is the average rectified integrated EMG ± 1 S.D. recorded during steady walking at natural speed (time 0: stance limb heel-strike); the middle trace (stopping) is the activity recorded at the termination of gait in response to a visual cue delivered at the heel-strike, and the lower trace (difference) is the net braking activity obtained from the above signals by subtraction. The corresponding average Rx profile and the ground contact (Gr. contact) phases of the stance limb during stop trials are reported at the bottom, on the same time scale. Averaging was synchronised on the first contact of the right (stance) limb with the platform. SOL, soleus muscle; QUAD, quadriceps muscle (vastus medialis); TA, tibialis anterior; HAM, medial hamstrings (semimembranosus–semitendinosus complex; E, E1, E2 and E3 are positive (excitatory) braking components.

referred to as the stance limb), and (2) later components on the side entering the swing phase during the same period (the left limb, hereafter referred to as the swing limb). A quantitative analysis of these actions is presented below.

Stance limb

Representative examples and spatio-temporal parameters of the positive braking activities isolated in the stance limb are reported in Fig. 2 and Table 1. In general, the plantar flexor SOL was the first muscle to present braking-related excitatory changes (SOL-E_{stance}). These started approximately 150 ms after the stop signal and consisted of significant enhancements of the desynchronised activity that restrains tibial advancement in the post-contact period during steady walking. In the antagonist TA, the positive actions included an earlier, less consistent, component recruited in mid-stance (TA-$E1_{stance}$) and a later component (TA-$E2_{stance}$), which anticipated the locomotor burst subserving foot clearance during the subsequent swing phase. Likewise, the QUAD muscle presented two positive components: the first (QUAD-$E1_{stance}$) was less frequent and was largely superimposed on the minor activity that precedes the toe-off during steady walking, and the second, more frequent component (QUAD-$E2_{stance}$) was activated in early swing, when the QUAD group is typically silent. In the posterior thigh HAM muscles there were usually three major braking peaks. The earliest (HAM-$E1_{stance}$) emerged in mid-stance and was normally slightly delayed with respect to SOL-E_{stance}; the intermediate component (HAM-$E2_{stance}$) coincided in part with the weak HAM activity associated with toe-off, and the last, most prominent component (HAM-$E3_{stance}$) anticipated the large HAM burst evoked prior to heel-contact during steady walking.

Figure 3 indicates that the average braking synergy in the weight-supporting limb primarily involved a distal-to-proximal activation of the posterior muscles of the leg (SOL, average onset time 13 % of the control stride) and thigh (HAM, average onset time 18 %). Positive activities were also isolated in the ES and GLM, with onset times at, respectively, 18 ± 3 and 35 ± 3 %. With the exception of SOL, which showed a single EMG burst, all of the muscles showed two or three progressively larger components organised reciprocally on the antagonistic groups. The earlier components (e.g. SOL-E_{stance} and HAM-$E1_{stance}$)

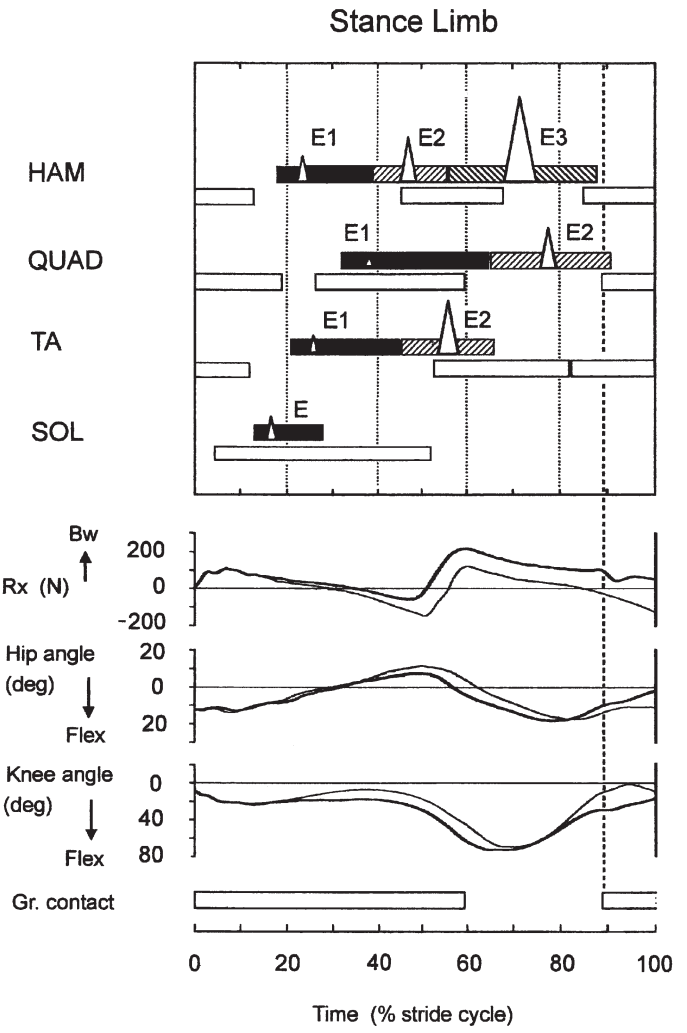

Figure 3. Organisation of the braking synergy and main biomechanical correlates in the stance limb

In the upper panel, the shaded boxes indicate the time periods occupied by the individual positive EMG components, normalised to a reference stride cycle measured during steady walking (time 0: stance limb heel-strike). The times of maximum amplitude of the individual components are marked by arrowheads, whose size is proportional to a consistency index related to the rate of occurrence, duration and peak amplitude of the component (see Methods). Open boxes indicate the period(s) of activity of each muscle during steady walking (low-level recruitment phases are included, such as HAM around toe-off and QUAD around heel-off). Data are averages of five subjects for the three walking speeds. The average Rx profiles, the sagittal hip and knee joint angles (0 deg: standing value) recorded during control (thin line) and stop trials (thick line), and the stance limb ground contact periods during stop trials are reported below, on the same time scale.

Table 1. Stance limb positive braking components

		Walking speed	Occurrence rate (%)	Absolute onset latency (ms)	Relative onset latency (%)	Relative peak latency (%)	Relative peak amplitude (%)
SOL	E	Slow	100	159 ± 30	12 ± 2	15 ± 2	69 ± 13
		Natural	100	146 ± 11	13 ± 1	18 ± 1	54 ± 29
		Fast	100	141 ± 14	15 ± 2	19 ± 1	24 ± 18
TA	E1	Slow	40	277 ± 24	20 ± 3	25 ± 5	53 ± 20
		Natural	60	228 ± 29	21 ± 5	27 ± 3	52 ± 36
		Fast	80	203 ± 19	23 ± 2	26 ± 3	51 ± 23
	E2	Slow	100	739 ± 190	53 ± 9	66 ± 9	63 ± 22
		Natural	100	487 ± 69	44 ± 6	57 ± 5	75 ± 42
		Fast	100	343 ± 56	36 ± 4	47 ± 4	97 ± 40
QUAD	E1	Slow	0	—	—	—	—
		Natural	60	355 ± 40	33 ± 5	41 ± 5	33 ± 26
		Fast	20	266*	26*	31*	61*
	E2	Slow	100	977 ± 99	69 ± 4	82 ± 3	55 ± 21
		Natural	80	684 ± 20	64 ± 4	80 ± 9	40 ± 26
		Fast	80	567 ± 124	63 ± 11	76 ± 8	48 ± 36
HAM	E1	Slow	60	348 ± 87	24 ± 4	32 ± 1	31 ± 20
		Natural	60	169 ± 30	16 ± 2	21 ± 1	55 ± 40
		Fast	80	139 ± 38	15 ± 2	22 ± 3	73 ± 37
	E2	Slow	100	582 ± 113	42 ± 5	48 ± 2	39 ± 28
		Natural	100	437 ± 58	40 ± 3	50 ± 3	65 ± 47
		Fast	80	311 ± 63	34 ± 3	44 ± 4	125 ± 78
	E3	Slow	100	820 ± 110	59 ± 4	74 ± 2	71 ± 19
		Natural	100	613 ± 48	56 ± 4	71 ± 6	100 ± 32
		Fast	100	485 ± 45	53 ± 3	73 ± 5	103 ± 17

For each walking speed (slow, natural and fast), data from the five subjects examined were pooled and mean ± S.D. values are reported for the spatio-temporal parameters. SOL, soleus muscle; TA, tibialis anterior muscle; QUAD, quadriceps (vastus medialis) muscle; HAM, medial hamstrings (semimembranosus–semitendinosus complex). E, E1, E2 and E3 are positive (excitatory) braking components. * Due to EMG artefacts, there were too few values to calculate the S.D.

were suitably located in time to play an exclusively functional braking role during the current stance phase, whereas components recruited at a longer latency were associated with the execution of the final controlled step to bring the right foot alongside the left one and, as such, cannot be considered pure braking actions.

The mechanical outcome of the early braking activities, quantified on the basis of the horizontal ground reaction forces (Rx), presented a moderate but reproducible enhancement of the backward-directed wave, which reflects the positive braking action exerted during weight acceptance during steady walking (Fig. 3). On average, the extra braking effect started 229 ± 41 ms after the stop signal, correlating well with the short-latency components in the EMGs, and always occupied the second half of the backward Rx wave (single support period), which during the gait termination trials became 12% longer (five subjects, three walking speeds; Student's t test for paired values, $P < 0.05$) and 20% larger ($P < 0.001$) than during steady walking. Over the same period, goniometric measurements indicated a slight increase in the knee flexion compared to the control values (Fig. 3).

As the walking speed rose from slow (mean 49 ± 8 per cent of the subject's height per second, % height s^{-1}), to natural (77 ± 7% height s^{-1}), to fast (120 ± 17% height s^{-1}), the main positive activities of the stance limb changed, but not uniformly, in that they were progressively anticipated and enhanced in the posterior proximal (HAM) group, but simultaneously depressed in the posterior distal (SOL) muscle (Table 1 and Fig. 4). In the HAM in particular, the onset times of the three components were significantly shortened (absolute latency *vs.* walking speed: E1, $r = -0.81$, $P < 0.005$; E2, $r = -0.87$, $P < 0.0001$; E3, $r = -0.91$, $P < 0.0001$; relative latency *vs.* walking speed: E1, $r = -0.69$, $P < 0.05$; E2, $r = -0.75$, $P < 0.005$; E3, $r = -0.63$, $P < 0.05$) and the relative duration and peak amplitude tended towards a positive correlation and showed a significant positive correlation with the walking speed, respectively (peak amplitude *vs.* walking speed: E1, $r = 0.71$, $P < 0.05$; E2, $r = 0.70$, $P < 0.005$; E3, $r = 0.54$, $P < 0.05$); a similar pattern was also observed in the ES and GLM.

By contrast with the proximal groups, the positive activity in the posterior distal muscles (SOL-E_{stance}) involved a slight velocity-dependent shortening of the

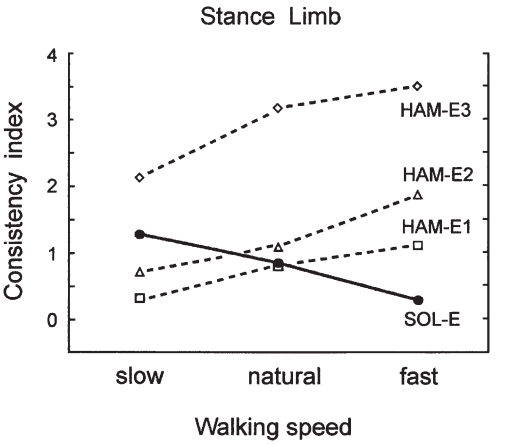

Figure 4. Velocity-dependent modulation of braking synergy in the stance limb, presented as changes in the consistency index of the main excitatory EMG components

Note the opposite behaviour of the posterior proximal (HAM) and posterior distal (SOL) muscle groups.

absolute onset latency which, because of the greater shortening of the stride time, resulted in a significant prolongation of the relative onset latency (latency *vs.* walking speed: $r = 0.76$, $P < 0.01$). In parallel, both the relative duration and peak amplitude were clearly reduced at higher walking speeds (duration *vs.* walking speed: $r = -0.70$, $P < 0.005$; peak amplitude *vs.* walking speed: $r = -0.80$, $P < 0.001$). This weakening was

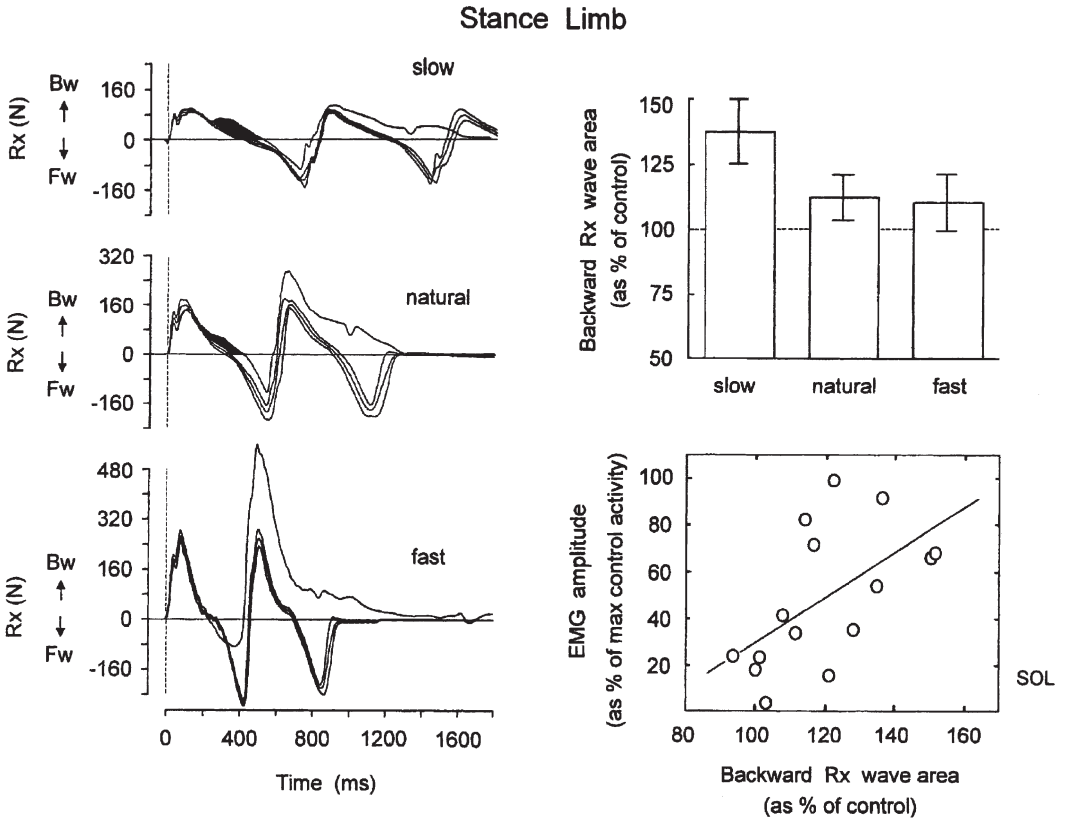

Figure 5. Velocity-dependent modulation of the mechanical braking effects in the stance limb

On the left, representative profiles of the horizontal ground reaction forces (Rx) recorded at the termination of gait for the three walking speeds are superimposed on the control templates obtained during steady walking (means ± 1 S.D.); the net braking effects on the earlier backward-directed wave are highlighted as filled areas. The upper histogram on the right reports, for the three walking speeds, the area of the earlier backward Rx wave in the stop trials, measured during the single-limb support periods and normalised to the control value (average of five subjects). The significant correlation between the area and the amplitude of the EMG braking component isolated in the ipsilateral SOL muscle (SOL-E_{stance}) is illustrated in the lower right-hand panel.

Table 2. Swing limb positive braking components

	Walking speed	Occurrence rate (%)	Absolute onset latency (ms)	Relative onset latency (%)	Relative peak latency (%)	Relative peak amplitude (%)
SOL	Slow	100	615 ± 87	44 ± 3	53 ± 4	29 ± 16
	Natural	100	430 ± 16	39 ± 2	54 ± 4	39 ± 16
	Fast	100	297 ± 68	32 ± 5	45 ± 2	59 ± 28
TA	Slow	0	—	—	—	—
	Natural	20	455 ± 52	42 ± 4	45 ± 1	57 ± 13
	Fast	80	331 ± 56	35 ± 3	42 ± 3	46 ± 18
QUAD	Slow	80	436 ± 46	31 ± 2	44 ± 1	48 ± 24
	Natural	100	345 ± 24	32 ± 2	45 ± 4	82 ± 40
	Fast	100	280 ± 35	30 ± 3	47 ± 5	114 ± 44
HAM	Slow	40	421 ± 33	30 ± 4	39 ± 4	53 ± 10
	Natural	40	328 ± 25	30 ± 4	40 ± 1	77 ± 40
	Fast	60	263 ± 24	29 ± 3	39 ± 5	108 ± 32

Same format as for Table 1.

confirmed by supplementary analysis of the integral of SOL-E_{stance} (mean 39 ± 20 arbitrary units, a.u., for slow; 39 ± 32 a.u. for natural; 18 ± 16 a.u. for fast speed; integral *vs.* walking speed, $r = -0.51$, $P < 0.05$).

These myoelectric changes were accompanied by distinct changes in the mechanical actions, in that the area of the extra-braking effects isolated in the backward Rx wave during the stop trials became proportionally smaller as

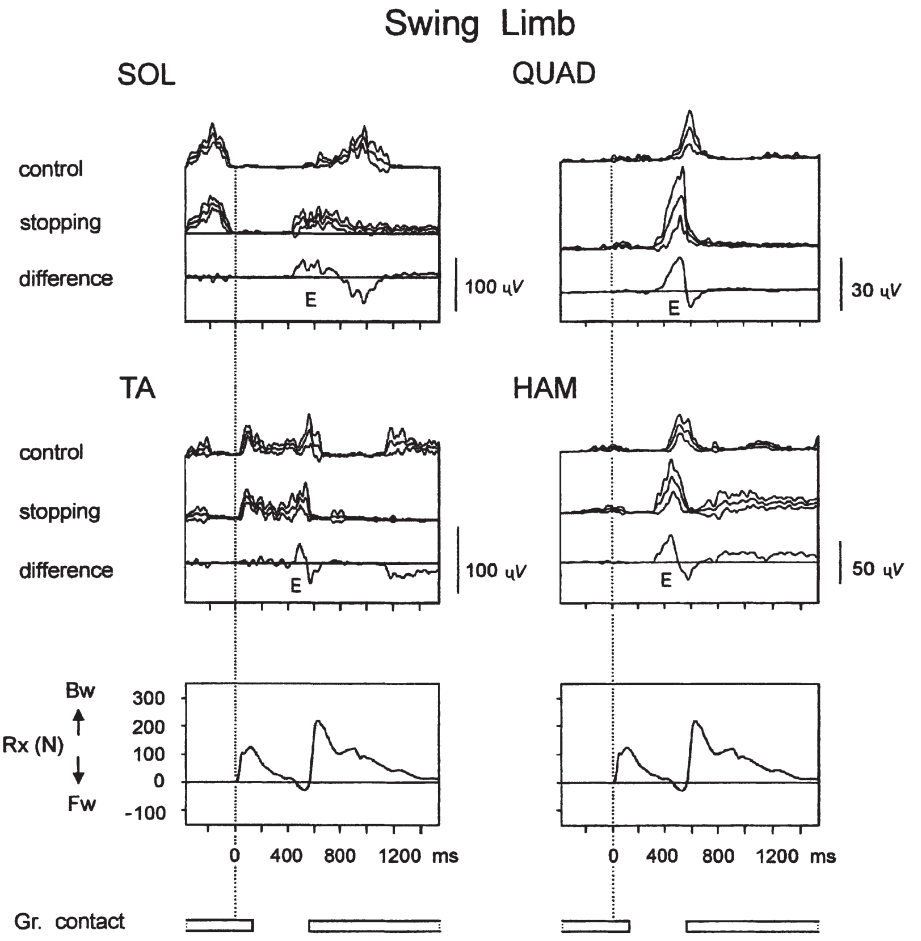

Figure 6. Examples of positive braking activities in antagonistic leg and thigh muscles of the swing limb

Data are from the same subject and are in the same format as for Fig. 2.

the walking speed rose (area *vs.* walking speed, $r = -0.70$, $P < 0.005$; Fig. 5). This reduction was significantly correlated with the velocity-dependent decline of the braking activity in the ipsilateral SOL muscle (e.g. area *vs.* peak amplitude of SOL-E_{stance}, $r = 0.58$, $P < 0.05$). Therefore, in keeping with the EMG findings, the early mechanical braking actions in the stance limb appeared to subside as walking became faster.

Swing limb

In the limb that was about to leave the ground at the appearance of the stop signal, the braking-related EMG changes had a mean latency of 330 ms and mostly covered the second half of the swing phase, before the final contact with the ground (Fig. 6 and Table 2). The individual action in the SOL (SOL-E_{swing}) consisted of anticipation and enhancement of the locomotor output which, in the control trials, was recruited during the subsequent weight-loading phase. In the TA, a minor activity with relatively low incidence (TA-E_{swing}) was superimposed on the rising portion of the EMG burst that normally precedes the heel-strike during steady-state walking. Similarly, in both the anterior and posterior thigh muscles, the command to stop induced premature recruitment of the main locomotor bursts initiated in the two groups before the ground contact, resulting in a synchronised positive activity in most of the trials in the QUAD (QUAD-E_{swing}) and about half the trials in the HAM (HAM-E_{swing}), with approximately the same onset latency.

Figure 7 shows that the representative thigh and leg muscles contributed to the swing limb braking synergy with single excitatory components that typically anticipated the locomotor output produced during steady walking. The braking actions always started proximally and constantly followed a proximal-to-distal sequence in which, according to our consistency criteria, the QUAD (average onset time 31 % of the control stride) and SOL (average onset time 38 %) played a prominent role (additional positive activities were seen in the ES and GLM, with onset times at, respectively, 17 ± 2 and 32 ± 2 %). The individual EMG components were largely co-activated on the antagonistic groups, especially in the thigh muscles, and showed close coupling of their onset latencies, which was absent on the contralateral side (QUAD-E_{swing} *vs.* HAM-E_{swing}, $r = 0.95$, $P < 0.01$; QUAD-E_{swing} *vs.* SOL-E_{swing}, $r = 0.88$, $P < 0.01$; and HAM-E_{swing} *vs.* SOL-E_{swing}, $r = 0.88$, $P < 0.01$).

The mechanical effects of these synergistic activities were measured on the backward Rx component produced

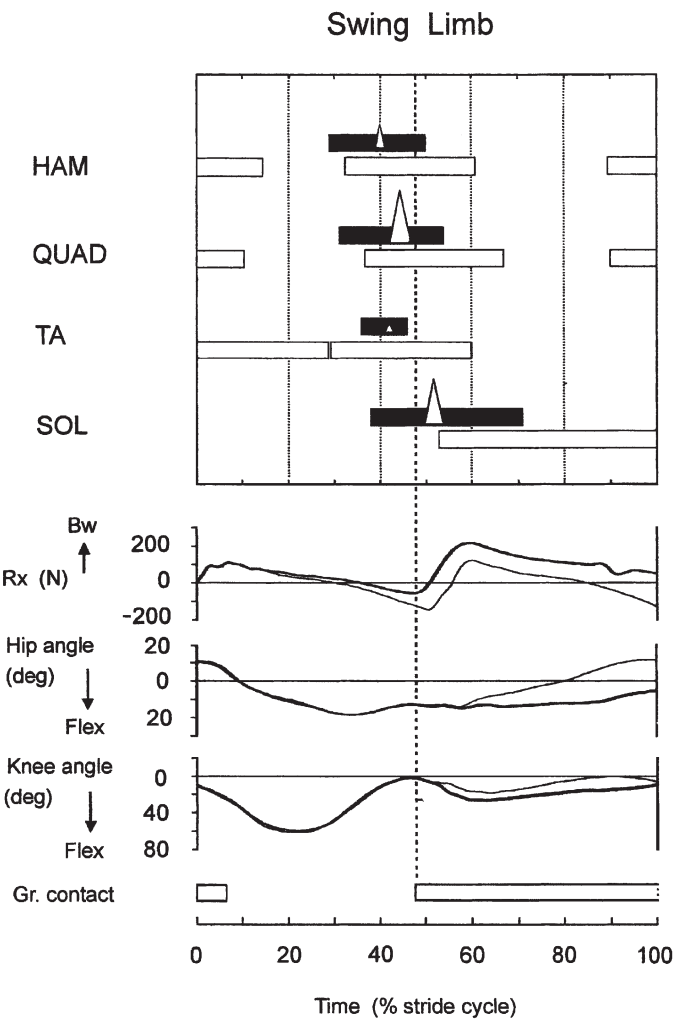

Figure 7. Organisation of the braking synergy and relevant biomechanical correlates in the swing limb

Time 0 indicates the delivery of the stop signal at the right (stance) limb ground contact instant, while the vertical dashed line marks the time of final contact with the ground of the left (swing) limb. Same format as for Fig. 3.

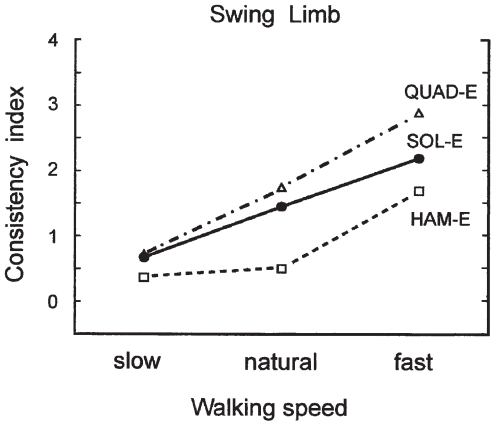

Figure 8. Velocity-dependent modulation of braking synergy in the swing limb, presented as changes in the consistency index of the main excitatory EMG components

Note the parallel behaviour of the proximal (QUAD, HAM) and posterior distal (SOL) muscle groups.

during the final contact of the left foot, which in the stop trials always occurred earlier than during steady walking (from 620 ± 57 to 560 ± 62 ms after the stop signal, five subjects, three walking speeds, Student's t test for paired values, $P < 0.01$). On average, the braking action assessed during the post-contact, single-support period, lasted 28 % longer ($P < 0.01$) and was as much as 370 % larger in area ($P < 0.01$) than the control values (Figs 7 and 9). Over the same time window, the hip and knee joint angles, which were not significantly modified at the ground contact time and immediately thereafter, showed slightly greater flexion before recovering towards their standing levels (Fig. 7).

With higher walking speed, the main braking components in the swing limb were stronger and were seen sooner (Table 2 and Fig. 8). In particular, the onset latency of SOL-E_{swing} was significantly shortened (absolute latency *vs.* walking speed, $r = -0.92$, $P < 0.0001$; relative latency *vs.* walking speed, $r = -0.83$, $P < 0.001$) and the relative duration and peak amplitude were greater (duration *vs.*

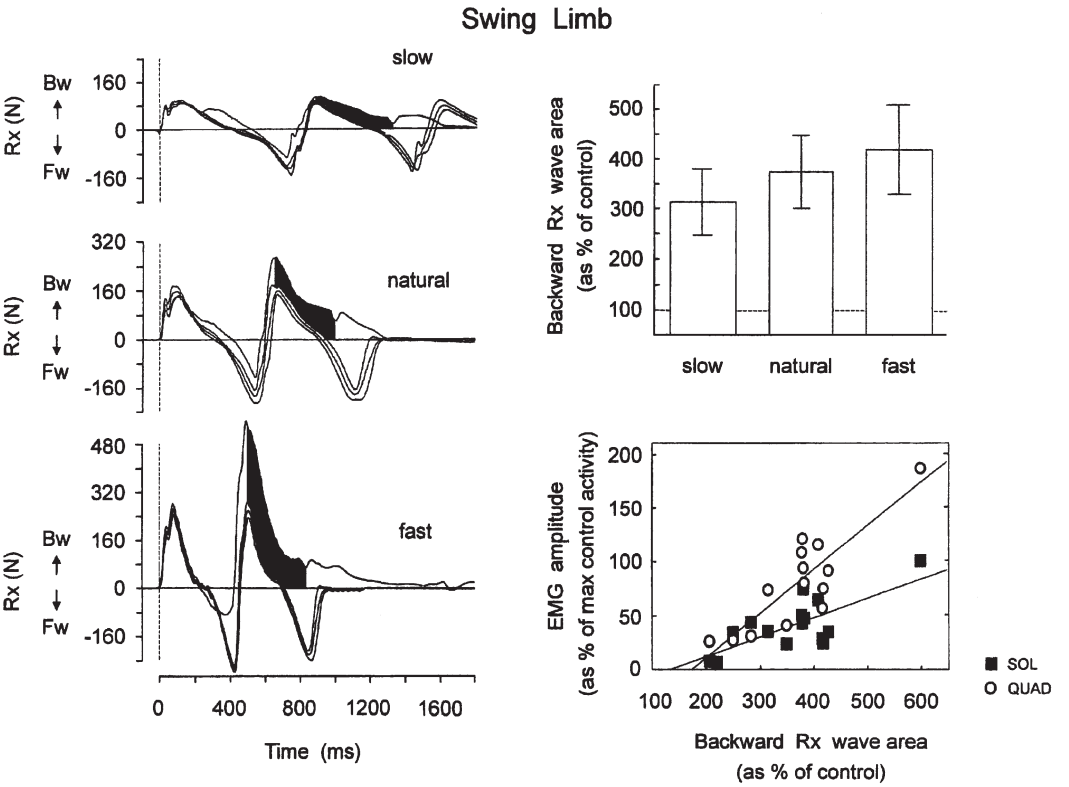

Figure 9. Velocity-dependent modulation of the mechanical braking effects in the swing limb

Same format as for Fig. 5. The upper histogram on the right side shows, for the three walking speeds, the area of the late backward Rx wave in the stop trials, measured during the single-limb support periods and normalised to the control value (average of five subjects). The lower right-hand panel shows the correlation between these areas and the amplitude of the EMG braking components isolated in the SOL (SOL-E_{swing}) and QUAD (QUAD-E_{swing}) muscles.

walking speed $r = 0.66$, $P < 0.01$; peak amplitude *vs.* walking speed $r = 0.64$, $P < 0.01$). Supplementary analysis of the EMG integrals confirmed the positive correlation between SOL-E_{swing} and walking speed ($r = 0.75$, $P < 0.005$; mean values 35 ± 27 a.u. for slow; 80 ± 28 a.u. for natural and 143 ± 75 a.u. for fast walking) and showed that the velocity-dependent increment of the background locomotor activity had a slope not significantly different from the stance side, where the homologous braking component (SOL-E_{stance}) was instead negatively correlated with the walking speed. The same anticipation and potentiation described for SOL also held for the QUAD (QUAD-E_{swing}, absolute onset latency *vs.* walking speed, $r = -0.86$, $P < 0.0001$, peak amplitude *vs.* walking speed, $r = 0.72$, $P < 0.005$; a similar pattern was also seen for ES and GLM).

In parallel with the EMG changes, the extra-braking actions isolated in the Rx wave when the left foot made final contact were significantly stronger when walking was faster (area during single support *vs.* walking speed: $r = 0.71$, $P < 0.05$). The close link between mechanical and myoelectric events was confirmed by the significant correlations between the area of the backward Rx wave and the amplitude of the positive braking components in the QUAD (QUAD-E_{swing}, $r = 0.85$, $P < 0.0001$) and SOL muscles (SOL-E_{swing}, $r = 0.74$, $P < 0.005$; Fig. 9).

DISCUSSION

The present study provides the first quantitative analysis of the rapid termination of gait in response to a visual cue, across a range of walking speeds. This motor task was examined in a single protocol, which required delivery of the halt signal at the instant of heel-strike, initial slowing down by the weight-bearing limb, stronger braking by the contralateral limb after its contact with the ground, and a final arrest with an extra step by the stance limb. As shown in pilot experiments (see Methods), the early braking actions in the stance leg and in the contralateral swing leg were observed even when subjects were asked to stop rapidly, with no particular instruction about how. Moreover, they were qualitatively similar regardless of whether subjects decided to stop in one step (i.e. with the stance leg backwards and the swing leg forward, which occurred in slow and natural-speed walking trials) or in one stride (i.e. making an extra step with the stance limb, as always occurred in fast walking trials). Within the limits of validity of a reactive behavioural condition, therefore, the early braking actions represent the basic components of a set of spontaneous stopping styles that are part of the natural repertoire (see also Hase & Stein, 1998).

Global organisation of the early braking actions in the weight-bearing and swinging limb

The braking synergy in the weight-bearing leg involved the activation of lower limb and posterior trunk muscles. In the leg and thigh the prominent role of the posterior muscles, SOL and HAM, is evidenced by the high consistency of their EMG profiles and by the compatible delay between their onset latency and the earliest changes in the backward Rx wave (80 ms on average for the SOL component). The presence of both of these braking activities at all three walking speeds indicates that even when only slow walking must be arrested, positive effects are still needed together with the expected withdrawal of the ongoing propulsive drive. According to several experimental observations, moreover, the coupled contraction of calf and HAM muscles occurs in various functional conditions which, like the termination of gait, mainly entail sagittal movements in the standing subject. For instance, it is the first EMG event observed during the forward sway induced by rapid displacements of the support platform (e.g. backward translation and toe-up rotation; Nashner *et al.* 1979) or by fast voluntary movements of the upper limbs (e.g. pulling on a handle), performed either while standing still (Cordo & Nashner, 1982) or during the weight-bearing period of the locomotor cycle (Nashner & Forssberg, 1986; Hirschfeld & Forssberg, 1991). Thus, a common set of muscle activities is triggered: (1) by peripheral signals as a compensatory reflex response to external destabilisation, (2) by central commands responsible for postural adjustments associated with fast intentional movements, and (3) by volitional commands for terminating gait. In this respect the triceps surae–HAM synergy can be regarded as a widely adopted motor programme available for starting deceleration of the advancing body by the weight-bearing limb.

As shown for the stance side, the positive activities isolated on the side entering the swing phase when the stop signal appeared involved both lower limb and posterior trunk muscles. Quantitative analysis of the EMGs for the thigh and leg groups, however, showed differences in organisation. First of all, the swing limb synergy consisted of single EMG components prevailing in the knee and ankle extensors, as documented by the high consistency of QUAD and SOL activities, and the correlation between their amplitudes and the size of the backward Rx waves during the ipsilateral final contact. Unlike on the stance side, moreover, where the distal muscles were usually activated first – probably in relation to the possibility of direct action on the ground reaction forces – the average recruitment order here was normally proximal to distal. In addition, the onset latencies of the braking activities were more closely correlated than on the opposite side, suggesting closer links between the motor commands to the individual muscle groups.

A further characteristic of the swing limb braking synergy concerned the activation periods of the EMG components in the antagonistic muscles, which showed remarkable similarities at both the proximal and distal levels. The activity in the QUAD, for instance, peaked before the ground contact, not after as during steady walking, thus

increasing the co-contraction with the antagonist HAM component. This recruitment mode should enhance the mechanical stiffness of the knee joint in view of the greater absorption of kinetic energy on the subsequent final impact with the ground. Kinematic measurements did in fact show persistent knee extension both at ground contact and during the early weight-acceptance phase, and an increment of knee yielding only after the QUAD burst was de-recruited. The swing limb braking synergy therefore appears to be controlled by a motor programme with a fairly robust structure, whose possible aim is to pre-set a mechanical condition of the lower extremity, leading to the maintenance of an extended configuration of the main bony segments and an increase in the mechanical impedance, in preparation for impact with the ground.

Interaction between the braking commands and the locomotor rhythm

Analysis of how the intentional commands interact with the locomotor pattern showed that, in several instances, the braking activities were produced during lengthening of the target muscles. The SOL-E_{stance}, for instance, was recruited during the lengthening contraction responsible for slowing the forward rotation of the shank in the weight-loading period, specifically during the phase of maximum lengthening velocity of the SOL muscle (from 10 to 20 % of the stride cycle; see Winter & Scott, 1991; Frigo *et al.* 1996). Similarly, the HAM-$E3_{swing}$ component attained its peak amplitude at between 70 and 75 % of the stride, when the degrees of current hip flexion (slightly more than the corresponding steady walking value, see Fig. 3) and knee extension (similar to the steady walking value) were compatible with stretching of the HAM group (Winter & Scott 1991; Schutte & Hayden, 1996). This braking action therefore presented a slight phase-lead with respect to the HAM lengthening contraction, which restrains the forward swing of the limb during steady walking, prior to ground contact.

Neurophysiological evidence suggests strongly that both the activation of the SOL during the weight-loading lengthening period and the contraction of the HAM during the swing phase lengthening period are associated with active (central) control of the velocity-sensitive excitatory inflow from the homonymous spindle afferents (Morin *et al.* 1982; Capaday & Stein, 1987; Crenna & Frigo, 1987; Crenna 1998; see also Taylor *et al.* 2000). Hence, one could surmise that the braking activities in these muscles at the termination of gait are started and/or enhanced by a centrally driven reinforcement of the autogenic excitation. In keeping with this hypothesis, it was shown recently that in spastic subjects, whose defective control of stretch reflex transmission is documented (e.g. Thillman *et al.* 1993), the locomotor lengthening contractions of the SOL and HAM muscles are increased in size and anticipated, in the presence of biomechanical evidence of enhanced braking actions well correlated in time with the abnormal EMG events (Crenna, 1998, 1999).

At variance with the homologous braking component on the contralateral side, the positive SOL-E_{swing} component started before touch-down, in a period of total EMG silence of the target muscle. During this stride phase, the excitability of the SOL motor nucleus is known to be low, most probably because of motoneurone (MN) hyperpolarisation, reciprocal inhibition associated with the TA heel-strike burst and pre-synaptic inhibition of the homonymous Ia input (see Crenna & Frigo, 1987). Therefore, in order to remove the inhibitory actions, any volitional command responsible for pre-contact braking activities will necessarily interfere with the duty cycle of basic segmental components of the locomotor rhythm generators. A relevant role of corticospinal systems in this motor control task is suggested by evidence that subjects with pyramidal lesions frequently show disturbances of the locomotor pattern, marked by ineffective inhibition of the SOL H-reflex in swing, and abnormal activation of the SOL muscle *before* the ground contact, two conditions associated with the restraint of forward progression during steady walking (Leonard *et al.* 1991; Crenna & Frigo, 1991*b*; Crenna & Inverno, 1994).

Velocity-dependent adaptation strategies

A notable finding of the present study was that although subjects always terminated gait in a similar way, the braking activities on the two sides were modulated differently as a function of walking speed. In fact, while a rather stereotyped pattern occurred in the swing limb (the positive components always scaling in parallel), on the stance side there were obvious differences in the proximal and distal muscles as walking became faster. The braking actions in HAM (and other hip and trunk muscles) were characterised by an increased occurrence rate, size and duration, whereas in distal muscles both the relative amplitude and duration of the braking actions became progressively less. This velocity-dependent depression cannot be accounted for by the non-linearity of EMG recruitment *vs.* background locomotor activity, because the same tendency was seen for the absolute size (integral) of SOL-E_{stance}. Neither is it likely to result from occlusion phenomena caused by locomotor excitation of progressively larger percentages of SOL MNs, because on the contralateral side a braking component of similar integral value (SOL-E_{swing}) was found to undergo velocity-dependent enhancement, in the presence of comparable increments of the locomotor output. The decrease in SOL activity, moreover, was accompanied by a parallel reduction of the mechanical effect in the relevant portion of the Rx profile. Thus, a negative modulation specific to the braking action of the SOL muscle appears to be produced in the stance limb when fast walking needs to be arrested.

A reasonable explanation may lie in the mechanical constraints associated with the weight-supporting phase

of the stride, specifically the velocity-dependent narrowing of the time window available for the production of deceleration by the foot plantar flexors. Indeed, we found that the relative onset latency of SOL-E_{stance} was significantly longer with faster walking speeds. As a consequence, any resulting mechanical effect will start progressively later in the support phase and, at the highest walking speeds, will eventually fall into the second half of the stance, when external forces (CM already ahead of CP) are no longer favourable for the production of braking actions (calf muscle activation in this period would have just the opposite effect: enhanced forward thrust). In this respect, when fast gait is to be arrested, lowering activity in the SOL and relative reinforcement in the antagonist TA becomes a biomechanical necessity. The same condition was reported by Hirschfeld & Forssberg (1991), who described a progressive decrease in calf muscle activity, time-coupled with voluntary arm pulls performed while walking, as the onset of the arm movement, and consequently calf muscle activity, was delayed in the stance phase. By the same reasoning, the velocity-dependent enhancement of motor output in the posterior thigh muscles (and also in the gluteal and paraspinal groups) simultaneously with SOL depression, might well be regarded as a form of compensation aimed at restraining the forward trunk flexion and slowing down the advancement of CM relative to CP.

We conclude that inherent mechanical constraints might dictate a divergence of individual muscle activities within the stance limb braking synergy, leading to the emergence of two different control strategies. The first, marked by a greater reliance on the posterior distal muscles, is adopted when the braking commands are issued in the early stance phase during slow and natural speed walking; the second strategy, involving a relative predominance of the posterior proximal muscles, is used when fast walking must be arrested. An important corollary of this notion is that information on the predicted position of CP *vs.* CM at the time the earliest braking commands will become mechanically effective must be continuously available in order to permit decision making and the execution of the appropriately tuned muscle synergy. This feed-forward adaptation most probably relies on integrated data from various populations of peripheral receptors with high dynamic sensitivity (i.e. providing higher-order derivatives of the variables measured as a basis for anticipating their values). However, it may also require internal models of the mechanical properties of the moving body in order to predict its forthcoming behaviour.

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